# Diagnosis and Treatment of Osteoarthritis

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**KEYWORDS**

• Osteoarthritis • Joint pain • Joint swelling • Joint inflammation • Osteophytes • Joint deformity

**KEY POINTS**

- Treatment options for osteoarthritis are generally based on symptom severity and duration, with the goals of symptom alleviation and improvement in functional status.
- Nonpharmacologic options include physical activity through land-based or aquatic exercises, acupuncture, transcutaneous electrical nerve stimulation, splints, and braces.
- Pharmacologic options are instituted in a stepwise approach, and include topical capsaicin, acetaminophen, nonsteroidal anti-inflammatories, cyclooxygenase-2 inhibitors, and intra-articular steroid injections.
- A surgical approach to osteoarthritis is reserved for chronic cases when pharmacologic and nonpharmacologic treatment options have already failed. Options include fusion and joint lavage, arthroscopy, and arthroplasty.

## BACKGROUND

Osteoarthritis (OA) refers to a heterogeneous group of conditions that lead to joint symptoms and signs associated with loss of integrity of the articular cartilage, in combination with changes in underlying bone and joint margins.[1] OA affects more than 40 million individuals in the United States alone, and is the leading cause of disability nationwide.[2] It is the most common articular disease worldwide, although frequencies vary by country.[3] The high prevalence of OA makes it one of the principal reasons for office visits in the primary care setting. OA causes with both direct and indirect economic costs to society. Clinician visits, medications, and surgical interventions comprise the direct costs, while comorbidities and time lost from work because of the effects of disability make up the indirect costs.[4] This situation is more evident among the elderly, who may lose their independence and may later need assistance with their daily living activities, thus adding to the economic burden.[5,6]

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OA can be subdivided into primary and secondary forms, with the primary, or idiopathic, form occurring in previously intact joints without any inciting agent.[4] Aging plays an integral part in this form of OA, as the wear and tear on the joints cause damage to the cartilage, leading to an abnormal repair mechanism. Certain diseases including primary generalized OA, erosive OA, and chondromalacia patellae are categorized as subsets of primary OA. The secondary form of OA is caused by an underlying predisposing factor, such as trauma (**Box 1**).

**Box 2** lists some of the risk factors that may predispose persons to develop OA. In general, any breach in the integrity of the chondrocyte matrix has the potential to cause OA.[2] Among these, obesity and joint injury are 2 of the strongest modifiable risk factors.[7] Hip OA has an important correlation with weight, genetic factors, sex, previous traumas, occupational factors, and age, whereas knee OA has a significant correlation with weight, lifestyle, and physical activity.[8]

OA develops by the combination of biochemical, cellular, and mechanical processes.[2] It is thought to start from the breakdown, by proteolysis, of the cartilage matrix. The weak matrix is prone to fibrillation and erosion, and results in the release of proteoglycans and collagen fragments into the synovial fluid. This process induces an inflammatory response in the synovium, which causes further cartilage degradation. As the cartilage becomes weak it begins to thin out, causing the joint space to narrow.[4] Damage to the cartilage also causes new bony outgrowths, or spurs, to form around the joints, which are evident on radiographs. The exact mechanism of pain generation in OA is not well understood, but is possibly related to an interplay of several mechanisms enumerated in **Box 3**.

## DIAGNOSIS

The diagnosis of OA is primarily based on thorough history and physical examination findings, with or without radiographic evidence.[9] Although some patients may be asymptomatic initially, the most common symptom is pain. Primary OA is usually symmetric and tends to initially affect the weight-bearing joints: the knees, hips, and spine. However, it is not uncommon for the joints of the hands and wrists to also become symptomatic. The pain is usually described as intense, deep, and "achy," worsened by movement or extensive use and relieved by rest and simple analgesics. Later on, as joints become more worn, the pain becomes more noticeable and unresponsive to medications. The pain causes reduction in range of motion and a decrease in

**Box 1**
**Secondary causes of osteoarthritis**

- Mechanical stress (obesity)
- Repeated trauma or surgery to the joint structures
- Infection
- Congenital abnormalities (abnormal joints at birth)
- Endocrine and metabolic disorders (diabetes, calcium deposition disorders)
- Other articular diseases (gout and pseudogout, rheumatoid arthritis)

*Data from* Lozada C. Osteoarthritis in Medscape reference. 2012. Available at: http://emedicine.medscape.com/article/330487-overview; and Hinton R, Moody RL, Davis AW, et al. Osteoarthritis: diagnosis and therapeutic considerations. Am Fam Physician 2002;65(5):841–9.

**Box 2**
**Risk factors for osteoarthritis**

- Age older than 50 years
- Obesity
- Trauma/injury to joints
- Genetics (significant family history)
- Reduced levels of sex hormones
- Muscle weakness
- Repetitive use (ie, jobs requiring heavy labor and bending)
- Infection
- Crystal deposition
- Acromegaly
- Previous inflammatory arthritis (eg, burnt-out rheumatoid arthritis)
- Heritable metabolic causes (eg, alkaptonuria, hemochromatosis, and Wilson disease)
- Hemoglobinopathies (eg, sickle cell disease and thalassemia)
- Neuropathic disorders leading to a Charcot joint (eg, syringomyelia, tabes dorsalis, and diabetes)
- Underlying morphologic risk factors (eg, congenital hip dislocation and slipped femoral capital epiphysis)
- Disorders of bone (eg, Paget disease and avascular necrosis)
- Previous surgical procedures (eg, meniscectomy)

*Data from* Lozada C. Osteoarthritis in Medscape reference. 2012. Available at: http://emedicine.medscape.com/article/330487-overview.

**Box 3**
**Osteoarthritic pain mechanisms**

- Osteophytic periosteal elevation
- Vascular congestion of subchondral bone, leading to increased intraosseous pressure
- Synovitis with activation of synovial membrane nociceptors
- Fatigue in muscles that cross the joint
- Overall joint contracture
- Joint effusion and stretching of the joint capsule
- Torn menisci
- Inflammation of periarticular bursae
- Periarticular muscle spasm
- Psychological factors
- Crepitus (a rough or "crunchy" sensation)
- Central pain sensitization

*Data from* Lozada C. Osteoarthritis in Medscape reference. 2012. Available at: http://emedicine.medscape.com/article/330487-overview.

functional capacity. Some patients feel stiffness that develops during rest, with morning joint stiffness for less than 30 minutes (morning stiffness longer than 30 minutes is more commonly associated with rheumatoid arthritis). Some may also report crepitus (a grating or cracking sensation) over the joint, which may or may not be associated with pain. Those with affected weight-bearing joints may exhibit an antalgic gait. Disease progression is characteristically slow, over several years or decades, causing the patient to become less active and more susceptible to morbidities associated with decreased physical activity, such as weight gain.[4]

Physical examination findings, when present, are mostly found on the affected joints. Most common are a reduced range of motion, crepitus, and intra-articular joint swelling, also called an effusion.[2] Sometimes malalignment and bone enlargement can be seen as well. Inflammatory changes, erythema, or warmth over the area are uncommon. These features are more likely to be seen in gouty or crystal arthropathies or in inflammatory arthritis, such as rheumatoid arthritis.[4] In late stages muscle atrophy around a severely affected joint can be seen. In OA of the hand, the distal interphalangeal (DIP), proximal interphalangeal (PIP), and trapeziometacarpal (base of the thumb) joints are affected. Heberden nodes and Bouchard nodes (**Fig. 1**), which are palpable osteophytic growths over the DIP and PIP joints, respectively, are more appreciable in women. In OA of the spine, associated changes are typically seen in the lumbar region, specifically the L3 through L5 levels. Facet arthritic changes cause foraminal narrowing, which may cause compression of the nerve roots. The later complication of lumbar spine OA is acquired spondylolisthesis.[4]

Inflammatory markers such as the erythrocyte sedimentation rate (ESR), C-reactive protein level, immunologic tests, and uric acid levels are typically within their reference range and usually do not need to be ordered, unless other conditions are being ruled out. No specific laboratory abnormalities are associated with OA.[2] Ancillary testing may be warranted if response to treatment is not as expected or the diagnosis remains uncertain. Synovial fluid is characteristically viscous and clear. Analysis usually shows a white blood cell (WBC) count of less than 2000/μL with mononuclear predominance,

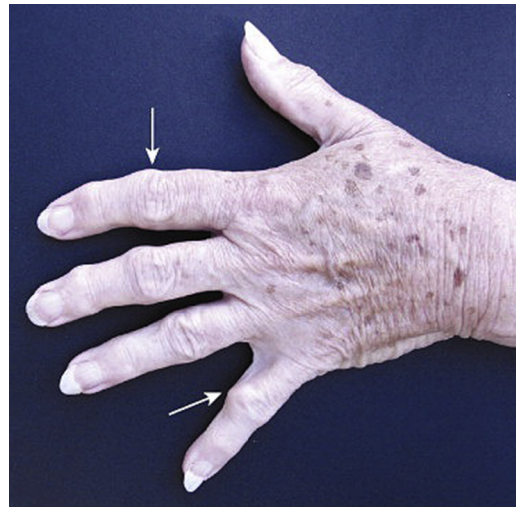

**Fig. 1.** Heberden nodes and Bouchard nodes (*arrows*). (*From* Waldman S. Physical diagnosis of pain: an atlas of signs and symptoms. 2nd edition. Philadelphia: Saunders; 2010; with permission.)

negative Gram stains and cultures, as well as the absence of crystals when the fluid is viewed under a polarized microscope. Ongoing research on the use of monoclonal antibodies, synovial fluid markers, and urinary pyridinium cross-links (ie, breakdown products of cartilage) as osteoarthritic indicators are under way. Discovery of a marker for early OA will aid in the diagnosis, monitoring, and targeted treatment of OA in the future.[4]

Plain radiography can help confirm the diagnosis, is readily available, and is cost-effective.[2] Typical findings are joint-space narrowing or loss, subchondral bony sclerosis, osteophyte formation, and cyst formation. **Figs. 2–4** illustrate these radiographic findings. Computed tomography (CT) or magnetic resonance imaging (MRI) is rarely used, unless other abnormalities are being ruled out. CT may be used to assist in the diagnosis of patellofemoral malalignment of the patellofemoral joint. Findings on MRI include chondral thinning, subchondral osseous changes, and osteophytes. In addition, direct visualization of the articular cartilage and other joint tissues (eg, meniscus, tendon, muscle, or effusion) is possible with MRI. Ultrasonography is currently being investigated as a tool for monitoring cartilage degeneration and for assistance with joint injections for treatment. Bone scans can help to differentiate OA from osteomyelitis and bone metastases, although these are not typically used in routine diagnosis.

## DIAGNOSIS: KEY POINTS

- OA refers to a heterogeneous group of conditions that lead to joint symptoms and signs associated with loss of integrity of the articular cartilage.
- The diagnosis of OA is primarily based on thorough history and physical examination findings, with or without radiographic evidence.

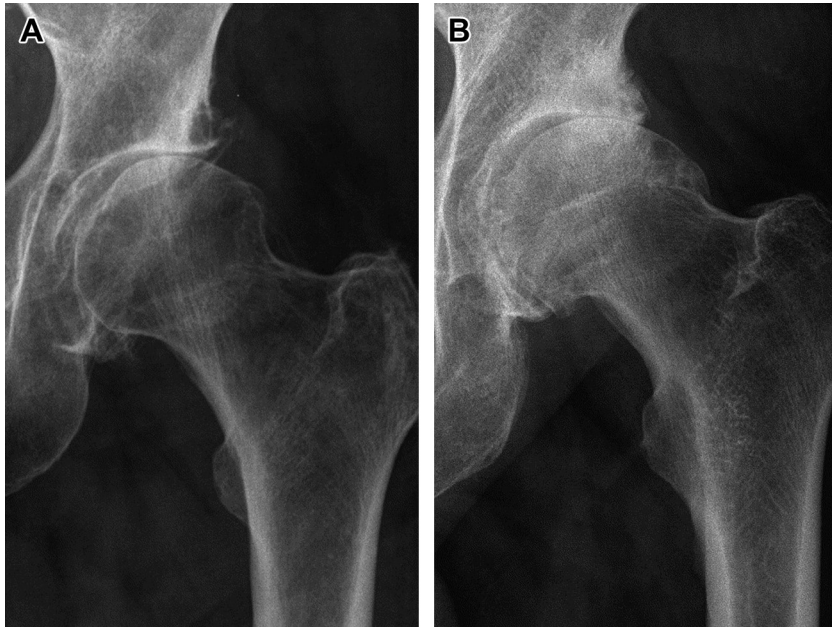


**Fig. 2.** Radiographs of hip osteoarthritis. (*From* Altman RD, Gold GE. Atlas of individual radiographic features in osteoarthritis, revised. Osteoarthritis Cartilage 2007;15(1):A1–A56; with permission.)

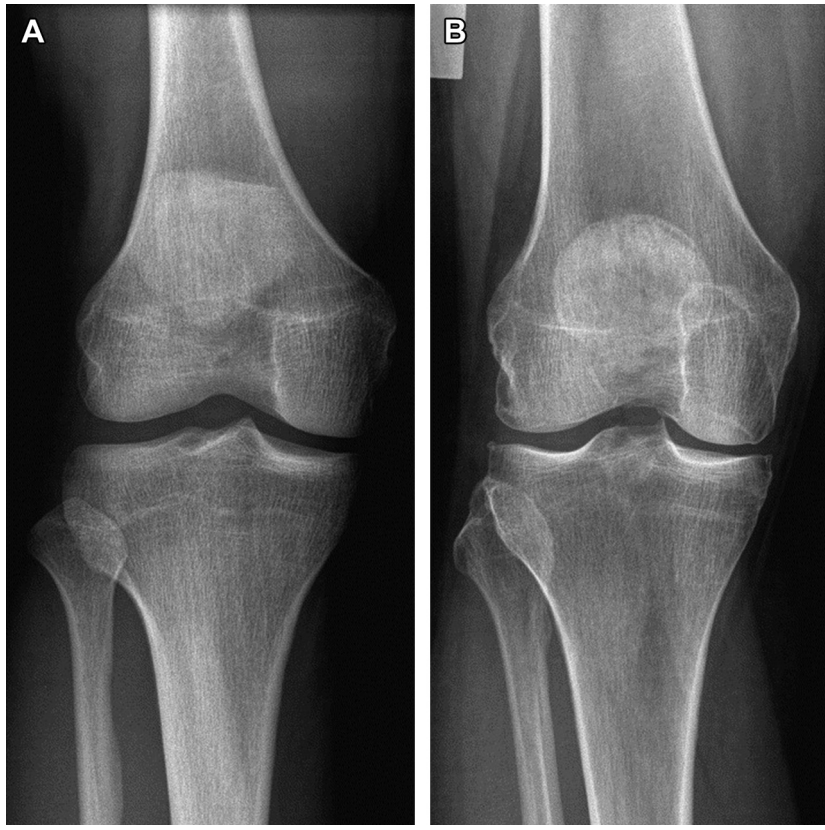


**Fig. 3.** Radiographs of knee osteoarthritis. (*From* Altman RD, Gold GE. Atlas of individual radiographic features in osteoarthritis, revised. Osteoarthritis Cartilage 2007;15(1):A1–A56; with permission.)

- The most common symptom is pain, described as intense, deep, and "achy," worsened by movement or extensive use and relieved by rest and simple analgesics.
- Most commonly affected are the weight-bearing joints: knees, hips, spine.
- Physical examination findings may include crepitus, effusion, decreased range of motion, and Heberden and Bouchard nodes in the hands.
- Typical radiographic findings include joint-space narrowing, subchondral sclerosis, osteophytic growths, and cysts.

## THE AMERICAN COLLEGE OF RHEUMATOLOGY CRITERIA FOR THE CLASSIFICATION AND REPORTING OF OSTEOARTHRITIS OF THE HAND, KNEE, AND HIP

### *Hand OA*

Patients are classified as having OA of the hand if they meet the criteria shown in **Box 4**. Sensitivity for hand OA if all of these criteria are fulfilled is 92%, and specificity is 98%. If at least 3 of these 4 criteria are met, sensitivity increases to 94% while specificity drops to 87%. Radiography was of less value than clinical examination in the classification of symptomatic OA of the hands. The 10 selected joints are the second and third DIP joints, second and third PIP joints, and the trapeziometacarpal joints of both hands.[10]

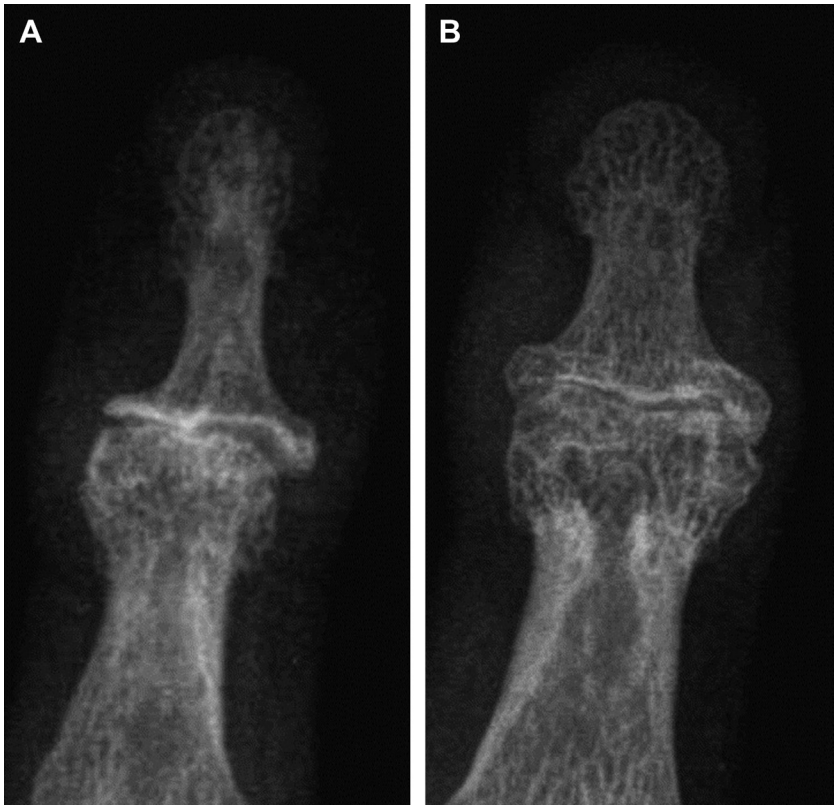


**Fig. 4.** Radiographs of hand osteoarthritis. (*From* Altman RD, Gold GE. Atlas of individual radiographic features in osteoarthritis, revised. Osteoarthritis Cartilage 2007;15(1):A1–A56; with permission.)

### *Knee OA*

A patient who has knee pain and at least 3 of 6 of the following is classified as having knee OA: age older than 50 years, stiffness of less than 30 minutes, crepitus, bony tenderness, bony enlargement, and no palpable warmth. Diagnosis based on these

**Box 4**
**Criteria for classification of idiopathic osteoarthritis of the hand**

History

- Hand pain, aching, or stiffness

Physical examination findings

- Hard tissue enlargement involving at least 2 of 10 selected joints
- Swelling of fewer than 3 metacarpophalangeal joints
- Hard tissue enlargement of at least 2 distal interphalangeal joints

*Data from* Altman R, Alarcon G, Appelrouth D, et al. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hand. Arthritis Rheum 1990;33(11):1601–10.

criteria was found to have a sensitivity of 95% and specificity of 69%. If laboratory findings are considered, an ESR of less than 40 mm/h and synovial fluid analysis are characteristic of OA (clear, viscous, WBC count <2000/mm$^3$); sensitivity with laboratory tests decreases to 92% but specificity increases to 75%. If radiographic findings of osteophytes are considered, the diagnosis is made with 91% sensitivity and 86% specificity (**Table 1**).[1]

### *Hip OA*

**Table 2** describes how hip OA may be diagnosed through clinical findings alone, and with radiographic findings.[11]

## TREATMENT

Treatment options for OA are generally classified as pharmacologic, nonpharmacologic, surgical, and complementary and/or alternative.[9] Typically, patients receive a combination of these treatment options to achieve optimal results.[12] Treatment initiation is based on symptom severity and duration, with the goals of symptom alleviation and improvement in functional status.[4] Individualization of treatment options is important.

### *Nonpharmacologic Modalities*

Physical activity has been widely proved to decrease pain and improve function in patients with OA.[13,14] The American College of Rheumatology (ACR) recommends both land-based and aquatic-based programs, depending on patients' comfort level and preferences.

Assistive devices such as walking canes, braces, and appropriate footwear may provide significant improvement in a patient's ability to perform activities of daily living (ADLs). Joint-protection and energy-conservation techniques must also be taught to

**Table 1**
**Criteria for classification of idiopathic osteoarthritis of the knee**

| Clinical | Clinical and Laboratory | Clinical and Radiographic |
|---|---|---|
| Knee pain | Knee pain | Knee pain |
| + | + | + |
| At least 3 of 6: | At least 5 of 9: | At least 1 of 3: |
| Age >50 y | Age >50 y | Age >50 y |
| Stiffness <30 min | Stiffness <30 min | Stiffness <30 min |
| Crepitus | Crepitus | Crepitus |
| Bony tenderness | Bony tenderness | + |
| Bony enlargement | Bony enlargement | Osteophytes |
| No palpable warmth | No palpable warmth | |
| | ESR <40 mm/h | |
| | RF <1:40 | |
| | SF signs of OA: clear viscous, or WBC <2000/mm$^3$ | |
| 95% sensitive | 92% sensitive | 91% sensitive |
| 69% specific | 75% specific | 86% specific |

*Abbreviations:* ESR, erythrocyte sedimentation rate; OA, osteoarthritis; RF, rheumatoid factor; SF, synovial fluid; WBC, white blood cell count.

*Data from* Altman R, Asch E, Bloch D, et al. Development of criteria for the classification and reporting of osteoarthritis: classification of osteoarthritis of the knee. Arthritis Rheum 1986;29(8):1039–49.

**Table 2**
**Criteria for classification of idiopathic osteoarthritis of the hip**

| Clinical | Clinical and Radiographic |
|---|---|
| 1. Hip internal rotation $\geq$15°, pain present on internal rotation of the hip, morning stiffness of the hip for <60 min, and age >50 y, or | Pain<br>+<br>At least 2 of the following criteria:<br>Osteophytes (femoral or acetabular) |
| 2. Hip internal rotation <15° and ESR $\leq$45 mm/h; if no ESR obtained, hip flexion $\leq$115° substituted | Joint space narrowing (superior, axial, and/or medial), and ESR <20 mm/h |
| 86% sensitive<br>75% specific | 89% sensitive<br>91% specific |

*Data from* Altman R, Alarcon G, Appelrouth D, et al. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hip. Arthritis Rheum 1991;34(5):505–14.

prevent further injury. For involvement of the trapeziometacarpal joint, the ACR recommends applying a splint.

Tai Chi is a form of Chinese martial arts consisting of slow, calculated movements, practiced for its health benefits and defense training. A pilot cluster-randomized trial among elderly patients with knee OA concluded that practicing Tai Chi can be efficacious in reducing pain and stiffness.[15]

Physical and occupational therapy are beneficial especially for deconditioned patients and postoperative patients who need to be retrained.

A pulsed electromagnetic field stimulation device and transcutaneous electrical nerve stimulation (TENS) are other possible treatment options for pain relief, both of which are thought to work by decreasing the pressure pain threshold.[16]

Acupuncture is another option for the treatment of chronic pain and physical dysfunction associated with OA.[4] Although data are limited, some studies have shown some benefit to acupuncture, especially when combined with pharmacologic treatment options,[17] although this therapy remains controversial.

Glucosamine sulfate (an amino-monosaccharide), glycosaminoglycans, and proteoglycans are substrates of hyaluronic acid, a major component of joint fluid.[2]

Glucosamine, chondroitin, and the 2 in combination have been the most extensively studied. Some improvement in pain and functional indices and a decrease in the loss of joint-space width have been demonstrated in some, but not all, studies,[18] and this therapy remains controversial.

Balneotherapy, also known as spa therapy or mineral baths, is also used to treat OA, although scientific evidence is weak because of methodological flaws in the studies that have shown efficacy.[19] However, in a randomized, controlled, single-blind study, improvement of pain and function, as well as the quality of life in patients with hand OA, have been demonstrated with balneotherapy combined with magnetotherapy.[20] However, this also remains controversial.

## ACR RECOMMENDATIONS FOR NONPHARMACOLOGIC TREATMENT MODALITIES FOR OSTEOARTHRITIS

Nonpharmacologic modalities such as modification of ADLs, joint-protection techniques, assistive devices, and thermal agents are conditionally recommended by the ACR for OA of the hand. In addition, patients with OA involving the trapeziometacarpal joint have been found to benefit from hand splints.[13]

For patients with OA of the knee, it is strongly recommended that they participate in cardiovascular (aerobic) and/or resistance land-based exercise, or aquatic exercise, depending on individual patient preference and safety. It is also strongly recommended for symptomatic, overweight patients to lose weight. Other treatment options include self-management programs, manual therapy in combination with supervised exercises, psychosocial interventions, medially directed patellar taping for patellofemoral OA, medially wedged insoles for those who have lateral compartment OA, laterally wedged subtalar strapped insoles for those who have medial compartment OA, thermal agents, walking aids as needed, and other exercise programs, such as Tai Chi. Traditional Chinese acupuncture and TENS are recommended only when the patient with knee OA has chronic moderate to severe pain and is a candidate for total knee arthroplasty, but either is unwilling to undergo the procedure, has comorbid medical conditions, is taking concomitant medications that lead to relative or absolute contraindications to surgery, or the surgeon is not comfortable recommending the procedure.

For the initial management of hip OA, as for knee OA, it is strongly recommended that patients participate in cardiovascular (aerobic) and/or resistance land-based exercises, or participate in aquatic exercises, depending on individual patient preference and safety. It is also strongly recommended that symptomatic, overweight patients lose weight. Other treatment options include self-management programs, manual therapy in combination with supervised exercises, psychosocial interventions, thermal agents, and walking aids as needed.

### *Pharmacologic Modalities*

Topical capsaicin cream should be considered for adjunctive treatment of focal joint pain.[2] Capsaicin is a component from chili peppers that produces warmth and works by desensitizing neurons by depleting substance P, a pain neurotransmitter. In elderly patients, capsaicin is recommended as first-line treatment of choice for hand OA, although caution is advised because potential serious adverse effects have been observed in clinical trials. Concerns exist that capsaicin-induced nerve desensitization is not fully reversible, and that its autonomic nerve effects may increase the risk of skin ulcers in diabetic patients.[21]

Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) are the mainstay for the treatment of OA, and cyclooxygenase-2 (COX-2) inhibitors may be used if the former are not well tolerated. The maximum dose of acetaminophen is 4 g/d, and patients must be advised about the potential for overdose and adverse effects, especially when they take over-the-counter medications that have acetaminophen as one of their components. All of these agents have potential gastrointestinal, hepatic, and cardiorenal adverse effects, which increase with dose and duration of treatment. Proton-pump inhibitors (PPIs) should always be considered with an NSAID and with a COX-2 inhibitor in patients at higher gastrointestinal risk.[22]

Intra-articular steroid injections are another treatment option. Corticosteroids work by anti-inflammatory and antinociceptive actions. Corticosteroids have been shown to decrease pain and symptoms associated with OA for up to 3 weeks.[23] However, they must not be administered more than 3 to 4 times per year.[2] Several randomized trials have proven the benefits of intra-articular steroid injections. Although response varies, pain relief and functional improvement can sometimes be obtained for up to 1 year after the injection.[24]

Viscosupplementation, the intra-articular injection of hyaluronic acid or its derivative, is currently approved by the Food and Drug Administration only for patients

with knee OA. There is conflicting evidence on its efficacy. Some evidence suggests symptomatic and functional improvement for up to 5 to 13 weeks,[25] whereas others have found minimal or nonexistent effects, Its use in treatment should be individualized because results from trials are not yet generalizable. Its main risk is local adverse reactions from the injection.[26]

### *Other Agents*

Several research studies have focused on other agents with potential for modifying disease progression or treatment of symptoms, but most of these studies still have inconclusive results. *S*-Adenosylmethionine (SAMe) has been found to have the potential to treat pain and improve functionality, although evidence remains equivocal.[26] Doxycycline is also being investigated for its possible disease-modifying properties and potential to slow cartilage degeneration.[27] Other agents such as strontium ranelate, an agent being used for the treatment of osteoporosis, are being studied for their potential benefit on symptoms of patients with knee OA.[28]

Treatment options using complementary and alternative medicine have gained widespread use among patients because of their ubiquitous presence. In one study topical creams, rubs, and ointments were most commonly used, followed by spiritual methods, alternative providers (such as chiropractors), nutritional supplements, and mind-body therapies.[29] A systematic review has concluded that there is no sufficient evidence to recommend any of the practitioner-based complementary therapies (biofeedback, magnet therapy, chiropractic) for the management of OA, but neither is there sufficient evidence to conclude that they are not effective.[30] In addition, it is important for primary care physicians to discuss with their patients the use of these alternative treatment options, especially when prescribing medications that can cause potential interactions.[30]

## ACR RECOMMENDATIONS FOR PHARMACOLOGIC TREATMENT MODALITIES FOR OSTEOARTHRITIS

Pharmacologic modalities recommended for the initial treatment of OA of the hand include either topical or oral NSAIDs, topical capsaicin, or tramadol. Owing to the lack of randomized controlled trials, the ACR has not recommended the use of intra-articular therapies, opioid analgesics, oral methotrexate, or sulfasalazine. Because of a lack of data, no recommendations have been given by the ACR regarding the use of hydroxychloroquine.[13]

For the initial management of knee OA patients may try acetaminophen, oral and topical NSAIDs, tramadol, and intra-articular corticosteroid injections, in a stepwise approach. For patients 75 years and older, topical, instead of oral, NSAIDs are preferred. Because of the lack of randomized controlled trials, the ACR has not recommended chondroitin sulfate, glucosamine, and topical capsaicin for the initial management of knee OA. The ACR has made no recommendations regarding the use of intra-articular hyaluronates, duloxetine, and opioid analgesics. For those with a history of symptomatic or complicated upper gastrointestinal ulcer, use of COX-2 selective inhibitors, or a nonselective NSAID in combination with a PPI, is recommended. In patients with symptomatic knee OA, those unresponsive to previously stated modalities, and those either unwilling to undergo surgery or with contraindications for total arthroplasty, opioid analgesics and duloxetine are possible treatment options. Contraindications to these pharmacologic modalities must be considered, and individual risks and benefits assessed, before initiation.

For the initial management of hip OA, recommendations are similar to those of knee OA, with the exception of the use of topical NSAIDs, intra-articular hyaluronate injections, duloxetine, and opioid analgesics, which are not recommended, as evidence from randomized controlled trial regarding their benefits or safety are insufficient at present. Opioid analgesics are a treatment option in cases of symptomatic hip OA unresponsive to aforementioned modalities when the patients are unwilling or are not candidates for total joint arthroplasty.

Table 3 summarizes the various management recommendations for hand, knee, and hip OA.

**Table 3**
**Nonpharmacologic and pharmacologic recommendations for the initial management of hand, knee, and hip osteoarthritis (OA)**

| Hand OA | Knee OA | Hip OA |
|---|---|---|
| **Nonpharmacologic** | | |
| Evaluation of ADLs<br>Joint protection techniques<br>Assistive devices, as needed<br>Thermal modalities<br>Splints (for trapeziometacarpal joint OA) | Aerobic/land-based exercise<br>Aquatic exercise<br>Weight loss<br>Self-management programs<br>Manual therapy in combination with supervised exercise<br>Psychosocial interventions<br>Medially directed patellar taping<br>Medially wedged subtalar strapped insoles (for those with lateral-compartment OA)<br>Laterally wedged subtalar strapped insoles (for those with medial-compartment OA)<br>Thermal agents<br>Walking aids, as needed<br>Tai Chi programs<br>Traditional Chinese acupuncture<br>Transcutaneous electrical stimulation | Aerobic/land-based exercise<br>Aquatic exercise<br>Weight loss<br>Self-management programs<br>Manual therapy in combination with supervised exercise<br>Psychosocial interventions<br>Thermal agents<br>Walking aids, as needed |
| **Pharmacologic** | | |
| Topical capsaicin<br>Topical NSAIDs, including trolamine salicylate<br>Oral NSAIDs, including COX-2 selective inhibitors<br>Tramadol<br>For persons aged $\geq$75 y, topical rather than oral NSAIDs | Acetaminophen<br>Oral NSAIDs<br>Topical NSAIDs<br>Tramadol<br>Intra-articular corticosteroid injections | Acetaminophen<br>Oral NSAIDs<br>Tramadol<br>Intra-articular corticosteroid injections |

*Abbreviations:* ADLs, activities of daily living; COX-2, cyclooxygenase-2; NSAIDs, nonsteroidal anti-inflammatory drugs.

*Data from* Hochberg M, Altman R, April KT, et al. American College of Rheumatology 2012: recommendations for the use of nonpharmacologic and pharmacologic therapies in osteoarthritis of the hand, hip, and knee. Arthritis Care Res 2012;64(4):465–74.

### *Surgical Approach*

If conservative treatment fails, surgical approaches to the treatment of OA can be considered. The most common indications for surgery are intractable pain and worsening disability.[9] Mechanical symptoms may also lead to surgical intervention. Surgical approaches to OA include fusion and joint lavage, osteotomy, arthroscopy, and arthroplasty.

### *Fusion and Joint Lavage*

Fusion, or the union of bones on either side of the affected joint, is a procedure done to relieve pain, usually when knee-replacement procedures fail or as an initial procedure for ankle or foot arthritis. However, this procedure puts more stress on the surrounding joints.[4] Joint lavage, although suggested in observational studies to give promising results, has failed to demonstrate pain relief or improvement of function in patients with knee OA.[31]

High tibial osteotomy is a procedure aimed at shifting the weight from the weakened cartilage on the medial aspect of the knee to the healthy lateral aspect of the knee, used primarily for younger patients with significant malalignment of the lower extremity, usually genu varum or bowleg deformity, and isolated medial compartment OA. It is thought to improve physical activity by decreasing pain, and often delays total knee replacement until about 10 years later.[32] However, total knee replacement is more difficult following this procedure.

Arthroscopy is a minimally invasive procedure used for removal of meniscal tears and debridement of loose articular cartilage. Randomized trials of arthroscopic debridement for OA of the knee have consistently failed to show an advantage over maximal medical therapy combined with physical therapy.[9] However, in some prospective consecutive series, most patients with knee OA associated with unstable cartilage or meniscal injuries have reported good to excellent symptomatic results at short-term and mid-term follow-ups after arthroscopy.[33] Therefore, arthroscopy is not recommended for nonspecific “cleaning of the knee” in OA, owing to its varying success rates.[4]

Arthroplasty is the surgical replacement of joint surface with a metal and plastic prosthesis, and is the treatment of choice for severe symptomatic OA. A variety of prosthetic devices are available, although studies are lacking regarding the advantage of one against the other.[9] Arthroplasty is performed if all other modalities are ineffective, if osteotomy is not appropriate, or if a patient cannot perform ADLs despite maximal use of the other treatment options already mentioned. This procedure alleviates pain and may improve function. Excellent patient outcomes following total joint replacement of the hip, knee, and shoulder have been reported.[9] Possible complications include infection and thrombophlebitis, with or without pulmonary embolism.[34] The use of perioperative antibiotics has decreased the incidence of postoperative infection, while early ambulation and administration of heparin or warfarin as prophylactic treatment for thrombosis are currently being observed. In the absence of complications, a minimum of 10 to 15 years of viability is expected after the procedure.[4]

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